EDITORIAL COMMENT*

THE RÔLE OF HISTAMIN IN ALLERGY

Sir Thomas Lewis,[1] in his researches upon the blood vessels of the human skin, predicated the hypothesis that the urticarial reaction of the skin to injury is due to the release of a histamin-like substance. With scientific caution, he has been chary in identifying this substance as histamin, despite the fact that practically all evidence points to this identity. However, the rôle of this histamin-like substance in the production of a characteristic allergic reaction such as urticaria has stimulated a search for further evidence linking the allergic and anaphylactic reactions to histamin.

Dale,[2] in his Croonian lectures, mentions the fact that chemical analysis of the lungs of man shows 35 milligrams of histamin per kilo, and the epidermis 24 milligrams per kilo. The significance of this lies in the fact that the lungs and epidermis of man contain the largest proportion of histamin, and are also the most common sites of allergic reactions.

Gebauer and his co-workers[3] have obtained a histamin-like substance from vena cava blood after anaphylactic shock. They feel that all evidence points to the identity of this substance with histamin, and probably to its identity with Manwaring's hepatic anaphylotoxin. Using strips of guinea-pig intestine for testing purposes, they found that vena cava blood after anaphylactic shock is active, while systemic and portal blood is not; and also that the substance is not specific and has no latent period in its action on the intestinal strip. They also show that the substance is easily diffusible, basic in character, inactivated by diazotized sulphanilic acid, lowers blood pressure on injection in cats, produces characteristic wheals, and does not contract the mouse intestine, all these properties being characteristic of histamin.

Bartosch, Feldberg, and Nagel[4] have found that the perfusion fluid from the anaphylactic guinea-pig lung also contains a histamin-like substance.

With these facts in mind, one is tempted to theorize on the possible rôle of histamin in clinical allergy: that in asthma there is a release of histamin in the lung, and in urticaria a release of histamin in the skin; that the low blood pressure commonly found in allergic individuals is a histamin effect. Although this theory affirms the mediation of histamin in the production of symptoms, we have as yet no acceptable explanation as to why histamin is released from the tissues, particularly in allergic individuals.

The field for speculation is wide and will undoubtedly stimulate research in the fundamentals of allergy; but as yet we must wait upon an explanation for the peculiarities of the allergic constitution.

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AMEBIC DYSENTERY

In the midst of so much discussion of amebic dysentery, it may be proper to call attention to one phase of a frequently recommended treatment.

The dosage of emetin hydrochlorid is often too large, when given over a short period of from nine to twelve days. It should be used in smaller dosage and extended over a longer period. Time is a vital element in the peculiar action of the drug. Large dosage will produce injury to the patient, and do but little damage to the amebae. We believe that one-third of a grain intravenously, daily the first week, repeated on alternate days thereafter for five doses, will accomplish as much as the large dosage and practically insure against injury by the drug. The same dosage twice weekly may be continued for months, with no cumulative action. The liver is thus protected and there is less danger of relapse. Meanwhile the treatment with carbarsone should start coincidentally with the emetin and be continued with one capsule each morning and evening, for ten days only. One should be constantly on guard against arsenical saturation, which is usually first manifested by redness or itching of the skin. The arsenical treatment may be repeated in full or in part, after a ten-day interval. One repetition usually suffices to rid the patient of tropozoites and cysts. Watchful search for organisms in the stools during the next year, at intervals of two or three months, is the safest procedure in guarding the patient against damage from the organism and from the drug.

Much has been said about the cumulative action of emetin and its damage to the heart muscle. There will be no heart symptoms or muscle tiredness, and none but good after-effects, if the above method is followed. The first complaint of muscle tiredness demands the lengthening of the intervals in the administration of the drug. This symptom shows saturation. The drug should not be given beyond its physiologic activity. The same is true of arsenic. This treatment, when so carried on, will save the profession much criticism and give the patient results equal to quinin in malaria. We have clinically checked the hearts of many emetin-treated patients and have been unable to find a single damaged heart, although many of these patients came complaining of heart symptoms at the beginning. Dr. Robert Langley has checked the electrocardiography of these hearts and has found every one normal.

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* This department of CALIFORNIA AND WESTERN MEDICINE presents editorial comment by contributing members on items of medical progress, science and practice, and on topics from recent medical books or journals. An invitation is extended to all members of the California and Nevada Medical Associations to submit brief editorial discussions suitable for publication in this department. No presentation should be over five hundred words in length.

1 Lewis, Thomas: Blood Vessels of the Human Skin. Shaw & Sons, Ltd., London, 1927.

2 Dale, H. H.: Croonian Lectures on Some Chemical Factors in the Control of the Circulation, Lancet, 216:1179, Vol. 1 (June 8), 1929.

3 Gebauer, E., Fuelnegg, E., and Dragstadt, C. A.: The Nature of Physiologically Active Substance Appearing During Anaphylactic Shock, Am. J. Physiol., 102:520-526 (Nov.), 1932.

4 Bartosch, R., Feldberg, W., and Nagel, E.: Weitere Versuche über das Freiwerden eines histaminähnlichen Stoffes aus der durchströmten Lunge sensibilisierter Meerschweinchen beim Auslösen einer anaphylaktischen Lungenstarre. Pflügers Archiv für die gesamte Physiologie des Menschen und der Tiere. 231. Band. 4. und 5. Heft. 1933.